The disturbance was sensory aphasia which usually lasted for about fourteen days. Then speech remained ungrammatical, patient was able to understand spoken language and had no difficulty in finding words. Only in the later stages of the disease did the amnestic aphasia become prominent and persistent. The capacity for repeating words was not markedly affected. After each apoplectic attack patient showed inability to read and understand what he read, but soon rapid improvement was noticed. At first complete agraphia was present, but he was soon able to write well. Later right-sided hemianopsia was demonstrated which often showed a slow and incomplete remission. After each attack of apoplexy a temporary paresis of the right side of the body was demonstrable. With the improvement of the paralysis a disturbance of purposeful execution of movements of the right upper and lower extremities was observed. For instance, patient made an attempt to put on a sock; after long manipulation he was not able to find the opening. However, he was successful when he used the left hand only. He took a handkerchief in the left hand and wiped his forehead correctly; he was asked to do the same thing with the right hand; he took the handkerchief between the third and fourth fingers, then in the first, but did not wipe the forehead. At last he threw it on the floor and wiped the forehead with the bare hand. He made false movements with the right leg in putting on trousers, etc. Such disturbances were occasionally seen on the left side, but they were only transient and not permanent. Remissions occurred on the right side, but they never completely disappeared. Impressions from external world were correctly conveyed. Stereognostic sense was at first slightly but later considerably affected.

The post-mortem examination of the brain revealed the following: Dura was adherent to the skull; marked cerebral arteriosclerosis, especially of the basilar arteries and arteria fossæ sylvii; the pia over the convexity was more adherent on the left than on the right side; no hemorrhagic areas could be demonstrated; the sulci of the left frontal lobe were widened, and the gyri were much reduced in volume; the left ascending parietal convolution, parietal and occipital lobe underwent great changes; also their convexities were rough and covered with a brownish membrane; the convexity of the left superior parietal lobe was affected and its superior convolution was narrowed; all the convolutions of the left occipital lobe were reduced in volume; the superior temporal gyrus of left temporal lobe was narrow, and also diminished in size and the surface was nodulated. The pathological condition of the encephalon is explained on the basis of arteriosclerosis.

M. J. KARPAS (Ward's Island).

The Journal of Mental Science

(Vol. LII. No. 217. April, 1906.)

1. Alcoholism, Crime and Insanity. W. BEVAN LEWIS.
2. Amentia and Dementia: A Clinico-Pathological Study. JOSEPH SHAW BOLTON.
3. The Morison Lectures for 1906. The Pathology of General Paralysis of the Insane. W. FORD ROBERTSON.
4. The Prognosis in Dementia Paralytica. GEORGE GREENE.

5. Mental (or Asylum-Trained) Nurses; Their Status and Registration. T. OUTTERSON WOOD.
6. The Nursing Staff at the Metropolitan Asylum, Leavesden; Notes Upon a Scheme of Promotion. FRANK ASHBY ELKINS.
7. On the Etiology of Asylum Dysentery. W. BERNARD KNOBEL.

1. *Alcohol, Crime and Insanity*—The author prefaces his remarks upon the relation of alcohol to crime and insanity, by a cursory review of the physiological effects of alcohol upon heat formation, deduced from animal experimentation, and the effects of alcohol upon the reaction time and muscular sense. He concludes that thermogenesis is modified in two ways by this agent; at first, there is a decreased thermogenesis due to the stimulating effect of the alcohol on the vaso-motor centers; this is followed by an increased heat production and radiation due to a paresis of these centers. He found, also, that chloral exhibited the same phenomenon to the conclusion that the incidence of alcoholism does not coincide with that of insanity, but does coincide with crime. These conclusions, which are somewhat at variance with other investigators, are based upon the statistics of different counties in England, where the social and racial conditions differ greatly and hence can be used for comparison. However, he does not belittle the influence of alcohol in the causation of insanity, not directly only, but through heredity. A very good exposition of the so-called laws of heredity is given. He doubts that alcoholic tendencies, as such, are inherited, but he is convinced that alcohol, like other toxic agencies in the parents, have a detrimental effect on the germ plasm. He deplores the loose term of "heredity" in cases of alcoholism, as well as in cases of toxemia—such, for example, as syphilis. Here a mother gives birth to a syphilitic child and the congenital syphilis may be the result of a simultaneous infection. That such conditions should be considered as the result of direct transmission rather than a hereditary influence, is a point that is well taken. The author goes even further into the analysis of the effects of these toxic agents on the germ plasm. He reasons from certain biological facts that there is a distinct difference between paternal and maternal forms of transmission to the embryo. The arrest of development, as shown in congenital mental weakness, idiocy and imbecility, he believes is the result of maternal toxemia, while convulsive neuroses, epilepsy, chorea, hysteria and like affections, are the legacy from the paternal side. This fact of alcoholism in the parents, especially at the time of conception, producing detrimental changes in the embryonic structures, has been considered by other authors, and is of considerable importance, and its relation to race hygiene should not be underestimated.

2. *Amentia and Dementia.*—Continued article.

3. *The Pathology of General Paralysis.*—This article is an abstract of the Morison Lectures, for 1906, published elsewhere, and gives the author's recent work upon the bacterial nature of general paralysis, and the relation of the diphtheroid bacillus, or *bacillus paralyticans,* as termed by him and his associates, to this disease. Various criticisms and reviews of the work have appeared, usually unfavorable to the view that general paralysis is caused by the diphtheroid bacillus. The criticism of Ferrier, in the Lumleian Lectures for 1906 (Royal College of Physicians, London), seems to be a fair and just estimate of this work by one of the author's own countrymen. While he considers the views of Ford Robertson interesting, he finds that there are many difficulties in the way of their general acceptance as

a satisfactory pathogeny of tabes, cerebral and spinal. Other investigators (Eyre and Flashman) found this bacillus in the throats of a large number of patients, suffering from different forms of insanity, as well as in the tissues of a considerable number of cases post-mortem, and there was no evidence that this bacillus was more common in the throats of general paralytics than of other insane patients. They were unable to trace any causal relation between diphtheroid bacilli and general paralysis. When competent bacteriologists review this subject, then the weak points of the theory are easily exposed. Dr. Bullock (quoted by Ferrier) states that the diphtheroid organism may be found in the intestines, genito-urinary tract, throat, and in the nasal secretions and elsewhere in perfectly normal individuals, as well as in catarrhal conditions of the respiratory tracts. He also states that the characters given this bacillus by Ford Robertson are not sufficient to differentiate it from other forms of diphtheroid organisms. If diphtheroid bacilli exist in the blood and cerebrospinal fluid of general paralytics, they should be capable of cultivation at once without waiting for the cessation of any supposed bactericidal action of these fluids, for no lysogenic action of the serum for bacilli of this group has been proved to exist. From specimens of cerebrospinal fluid, blood, and urine of several cases of tabes, and general paralysis, absolutely sterile cultures were obtained by competent bacteriologists for Ferrier, and he comes to the conclusion that it is not improbable that the bacilli found by Ford Robertson, so largely diffused throughout the tissues in general paralysis, are the result of a terminal invasion. This view seems perfectly justifiable. He also claims that it has not been shown that symptoms and post-mortem appearances of general paralysis can be produced by toxines prepared from these bacilli. It is difficult to see why it is necessary to have such a severe disease as syphilis in order to cause an impairment of the resistive power of the various tracts and thus allow the *bacillus paralyticans* to gain an entrance to the system; such an impairment could certainly be produced by a large number of conditions, and it seems unnecessary to bring syphilis into the etiology at all.

4. *The Prognosis in Dementia Paralytica.*—The conclusions of the author are based upon the analysis of 500 cases of general paralysis, and are interesting as showing what factors tend to limit or prolong the course of the disease. Among the factors that favor a long course are: *Youth,* from the age of 25 to 35, the duration of the disease steadily diminishes, and from the age of 35 to 40 the acutest form of the disease appears. (2) *Females* live nearly twice as long as males. (3) Recently *acquired alcoholism* has a tendency toward prolonging the course of the disease. He found the early onset of dementia a very unfavorable symptom, and that melancholic subjects rarely survive a year. In another paragraph, however, he states that melancholic types tend to remissions. Maniacal exalted types run a variable course. Systematized and fixed delusions indicate that the course of the disease will be protracted. Pupils inactive to light, sluggish reaction to light, rapidly alternating pupils are unfavorable signs. It is interesting to see that epileptiform convulsions have little or no general bearing on the course of the disease, and that the degenerate types live longer than the more highly developed subjects. The disease in juvenile subjects pursues a long and chronic course, and finally the association of early tabes with early general paralysis is an indication that the course of the latter disease is likely to be lengthened.

5. *Trained Nurses.*—Not suitable for abstracting.

6. *Nursing Staff.*—Not suitable for abstracting.

7. *On the Etiology of Asylum Dysentery.*—The author has endeavored by a thorough investigation to ascertain the cause for the prevalence of this disease in the English asylums. From the reports it would seem that dysentery is much more prevalent in the English asylums than in this country where happily it is rather rare. Scattered cases may occur with us, but never in such numbers as reported by English writers. He comes to the following conclusions: That dysentery in England is mainly confined to insane asylums, and does not occur except rarely in other large institutions, such as prisons and workhouses; that the increased precautions that have been taken during the past few years have made no appreciable difference to the incidence of or the mortality from this disease; that there is strong evidence in favor of the view that not one, but many micro-organisms, either singly or as a mixed infection, can give rise to dysentery. From the fact that dysentery develops after a disturbance of the subsoil in the neighborhood of an asylum, he argues that there must be some relation between this disturbance and the disease. That the evidence deduced from the relation between the inhalation of sewage effluvia and dysentery supports the theory that asylum dysentery can be caused by some microorganism which normally inhabits the colon and becomes pathogenic when the resisting power of the tissue is sufficiently reduced. That the occurrence of dysentery in members of the staff of an asylum is probably due either to infection by a virulent form of some universal organism, or to some normal colon organism becoming pathogenic, owing to the reduction of immunity caused by the frequent breathing of an atmosphere permeated by a fecal odor. That there is strong evidence to support the theory that, in the insane, the vitality and the resisting power of all tissues to infection is reduced, owing to the impairment of their trophic nerve supply. That dysentery is particularly apt to occur in the insane owing to the deterioration of nerve cells affecting the trophic nerve supply to the colon. That it is far less apt to occur in congenital cases of insanity or those in whom the mental disease is stationary. That the statistical evidence is entirely against the view that dysentery is spread by the transfer of recovered cases from ward to ward. He calls especial attention to the fact that it is hardly possible that the disease is communicated from one person to another, thereby disagreeing with Mott, who, in an earlier article on the subject, claimed the spread of dysentery was due to imperfect precautions against infection, but with the best possible means of precaution in Claybury Asylum had failed to lessen the mortality of the disease. H. H. COTTON (Hathorne, Mass.).

Review of Neurology and Psychiatry

(Vol. V. No. 3, 1907.)

1. Tabetic Atrophy of the Auditory Nerve. GOWERS.
2. Alcoholic Psychoses: A Study of Their Mechanism and of Their Relation to Other Mental Disorders. C. W. CHAPIN.
3. Dementia Præcox. J. MACPHERSON.

1. *Tabetic Atrophy of the Auditory Nerve.*—Gowers suggests that a progressive reduction of hearing from above and below, analogous to